

NeuroCOVID: Pathophysiology and neurological manifestation

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Abstract

At the end of 2019, the appearance of pneumonia of unknown etiology was reported. Later, it was confirmed that it was caused by a new coronavirus called SARS-CoV-2. Almost a year after being declared a pandemic, affecting millions of people worldwide, causing serious illness and death, the number of reports of neurological manifestations is increasing. Both as a form of presentation and during the evolution of the infection, cases of cerebrovascular disease, toxic metabolic encephalopathy, encephalitis, delirium and Guillain Berré syndrome have been reported. Experimental studies have corroborated the neurotropism of coronaviruses, assuming the pathophysiology from studies with SARS-CoV and MERS-CoV, since the genomic analysis of these is similar to SARS-CoV-2. Not only is the direct action of the virus on nerve cells postulated, through interaction with ACE receptors, but also the lesions generated by hypoxia, secondary to pulmonary involvement, the inflammatory response that awakens the cytokine cascade and thrombotic events since it has been seen to generate a procoagulant state. Published information has been compiled, pending epidemiological studies on a larger scale.)

Biography:

Leandro Tumino has received his medical degree from the Faculty of Medical Sciences of the National University of La Plata. He completed his Intensive Care residency at HIGA San Martín de La Plata, where he currently works as a physician in the intensive care unit and instructor for residents. He is director of the Committee of Neurocritical Care of the Argentine Society of Intensive Care.

Recent publication data:

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2. Moore ZE, Cowman S. Risk assessment tools for the prevention of pressure ulcers. *Cochrane Database Syst Rev.* 2014;(2):CD006471.
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5. McInnes E, Jammali-Blasi A, Bell-Syer SE, Dumville JC, Middleton V, Cullum N. Support surfaces for pressure ulcer prevention. *Cochrane Database Syst Rev.* 2015;(9):CD001735.
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